Pericardial Effusion After Cardiac Surgery: Risk Factors, Patient Profiles, and Contemporary Management

Elena A. Ashikhmina, MD, Hartzell V. Schaff, MD, Lawrence J. Sinak, MD, Zhuo Li, MS, Joseph A. Dearani, MD, Rakesh M. Suri, MD, DPhil, Soon J. Park, MD, Thomas A. Orszulak, MD, and Thoralf M. Sundt III, MD

Divisions of Cardiovascular Surgery and Biomedical Statistics and Informatics, Mayo Clinic, Rochester, Minnesota

Background. We aimed to review recent experience at our institution in the diagnosis and treatment of pericardial effusion after cardiac surgery and to identify risk factors for its development.

Methods. We searched our clinical database for patients 18 years or older who had cardiac surgery with cardiopulmonary bypass from 1993 through 2005. For patients with pericardial effusion (study group), medical records were reviewed to evaluate its manifestations and management. To identify risk factors for effusion, study patients were compared with patients without effusions. A second analysis compared the study group with a cohort without effusions who had routine postoperative echocardiographic examination.

Results. Of 21,416 patients identified, 327 (1.5%) had pericardial effusion (study group), 280 (86%) of whom had nonspecific symptoms. Clinical features of tamponade were documented in 138 patients (42%). Effusions were evacuated by echocardiography-guided pericardiocentesis (n = 169, 52%) or surgical drainage (n = 75, 23%). Effusion resolved after left thoracocentesis for pleural effusion in 3 patients (1%); 67 patients (20%) were treated conservatively. In 13 cases (4%), recurrent effusion required drainage after initial pericardiocentesis. Independent risk factors for effusion were larger body surface area, pulmonary thromboembolism, hypertension, immunosuppression, renal failure, urgency of operation, cardiac operation other than coronary artery bypass grafting, and prolonged cardiopulmonary bypass. Previous cardiac operations were associated with lower risk of effusion.

Conclusions. In our study, pericardial effusion occurred in 1.5% of patients, and symptoms were nonspecific. Several factors, mainly related to preoperative characteristics and type of operation, predispose patients to effusion. Echocardiography-guided pericardiocentesis is effective and safe in these patients.

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Despite many recent improvements in intraoperative management and postoperative care, late pericardial effusions remain an important cause of morbidity after cardiac surgery. Pericardial effusions may delay recovery and can be life threatening when tamponade leads to hemodynamic compromise [1]. In the modern era, because of widespread use of chronic anticoagulation and increased complexity of operations, the incidence of effusion may be higher [2]. Thus, it is important to have updated information about the manifestations, risk factors, and natural history of pericardial effusion to guide protocols for its prevention, diagnosis, and treatment. The objectives of this study were to review recent experience at our institution in the management of pericardial effusion, to assess modalities and outcomes of treatment, and to identify causative risk factors.

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Address correspondence to Dr Schaff, Division of Cardiovascular Surgery, Mayo Clinic, 200 First St SW, Rochester, MN 55905; e-mail: schaff@mayo.edu.

Patients and Methods

After approval by the Mayo Clinic Institutional Review Board (April 10, 2007), we searched our patient database for the records of patients aged 18 years or older who underwent cardiac surgery with cardiopulmonary bypass at Mayo Clinic, Rochester, MN, from 1993 through 2005. Those who died on the day of operation were excluded. From this group we identified the study cohort, which consisted of patients in whom pericardial effusion developed within 30 postoperative days. Pericardial effusion was defined as fluid in the pericardial space that manifested with symptoms, required specific treatment, was large enough to prompt consideration of drainage, or required lengthened in-hospital stay for observation. Patients who did not meet the above criteria, required reexploration for bleeding or tamponade within the first 3 days after surgery, or did not provide research authorization were excluded.

All other patients presumably did not have pericardial effusion after the operation and formed the first control group (control 1). Because not all of these control patients had routine postoperative echocardiographic examina-

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Table 1. Subject Variables Included Into the Univariate Analysis of Risk Factors for Pericardial Effusion

Patient Variables	Comorbid Conditions	Operative Factors
Age	Diabetes mellitus	Operative category (valve operation, coronary artery bypass grafting, heart transplant, aortic aneurysm surgery, other)
Sex	Hypertension	
Year of surgery	Hypercholesterolemia	
Body surface area	Renal failure	Status of operation (elective, urgent, emergent)
Body mass index	Infectious endocarditis	Use of intraaortic balloon pump
Weight	Immunosuppressive therapy	Ventricular assist device implantation
Height	Prior operations with or without cardiopulmonary bypass	Cardiopulmonary bypass time
New York Heart Association class	Myocardial infarction	Aortic cross-clamping time
Left ventricular ejection fraction	Cardiac heart failure	
Cardiac output	Cardiogenic shock	
	Chronic corticosteroid use before operation	
	Diagnoses (coronary artery disease, congenital pathology, valve pathology, aortic aneurysm, hypertrophic cardiomyopathy, left ventricular aneurysm, pulmonary thromboembolism)	

tions, some effusions, especially if asymptomatic, could have been underdiagnosed. Therefore, to decrease the number of false-negative cases among control patients, we identified a subset of the control patients who had at least 1 echocardiographic examination after 3 postoperative days (control 2).

We reviewed the medical records of all patients to obtain demographic characteristics, comorbid conditions, results of physical and echocardiographic examinations, chest radiograms, methods of treatment, and early clinical and echocardiographic follow-up (30 postoperative days). Of note, echocardiography either was part of a standard protocol of postoperative management or was performed because of suspicion of pericardial effusion on the basis of clinical symptoms.

Evidence of tamponade secondary to pericardial effusion was determined echocardiographically. Two-dimensional (2D) echocardiographic characteristics indicating tamponade were early diastolic collapse of the right ventricle, compression of heart chambers, plethora of inferior vena cava, or ventricular interdependence (eg, inspiratory interventricular septal shift); Doppler characteristics were respiratory variation of flow velocity across atrioventricular valves or in the hepatic vein.

Statistical Analysis

Descriptive statistics for categoric variables are reported as frequency and percentage; continuous variables are reported as mean (SD). Categoric variables were compared using the χ^2 test. Continuous variables were compared using the 2-sample *t* test or Wilcoxon rank sum test as appropriate.

Two separate analyses were performed. Predictors of pericardial effusion were determined first using all qual-

Table 2. Subject Characteristics

Characteristic	Group[a]				
	Patients With Effusions (n = 327)	Control 1[b] (n = 19,632)		Control 2[c] (n = 10,158)	
		Value	*p*[d]	Value	*p*[d]
Age, years	62 (14)	65 (13)	<0.001	64 (14)	0.003
Men	224 (69)	13,265 (68)	0.72	6,298 (62)	0.02
Body surface area, m^2	2.0 (0.3)	1.9 (0.2)	<0.001	1.9 (0.2)	<0.001
Body mass index, kg/m^2	29.2 (5.9)	28.3 (5.4)	0.01	27.8 (5.5)	<0.001
Heart pathology					
CAD	159 (49)	13,328 (68)	<0.001	5,130 (51)	0.89
Congenital	45 (14)	1,750 (9)	0.002	1,437 (14)	0.84
Valve	272 (83)	12,712 (65)	<0.001	9,094 (90)	<0.001
Aortic aneurysm	46 (14)	1,620 (8)	<0.001	972 (10)	0.007
HOCM	8 (2)	633 (3)	0.43	611 (6)	0.007
Prior operations using CPB	29 (9)	2,285 (12)	0.12	1,551 (15)	0.001
CPB time, minutes	112 (60)	90 (44)	<0.001	89 (45)	<0.001
Cross-clamping time, minutes	72 (40)	56 (30)	<0.001	60 (34)	<0.001
Preoperative EF	0.55 (0.17)	0.57 (0.14)	0.35	0.57 (0.15)	0.07
Intraaortic balloon pump	34 (10)	1,118 (6)	<0.001	615 (6)	<0.001

[a] Values are mean (SD) or no. of patients (%). [b] Patients without effusion. [c] Patients without effusion and available postoperative echocardiographic examination. [d] Comparison with study group (patients with effusions).

CAD = coronary artery disease; CPB = cardiopulmonary bypass; EF = ejection fraction; HOCM = hypertrophic obstructive cardiomyopathy.

ified patients without effusion as controls (control 1) and second, using patients without effusion who had postoperative echocardiographic evaluation from 3 to 30 days after surgery as controls (control 2). Logistic regression models were used to determine univariate and multivariate predictors of pericardial effusion. The variables included in the analysis are listed in Table 1. The multivariate model considered univariately significant variables ($p < 0.05$) with model selection using the stepwise method. Area under the curve and the Hosmer-Lemeshow test were used to assess the goodness-of-fit of the models. A bootstrapping method was used to assist in variable selections in the final multivariate model. All statistical tests were 2-sided with the alpha level set at .05 for statistical significance.

Results

Patients

Search of the database identified 21,416 patients at least 18 years old who underwent cardiac surgery with cardiopulmonary bypass during the study period; 162 patients died on the day of operation and were excluded. Other patients excluded were 860 reoperated for bleeding within 3 postoperative days and 435 without research authorization. Pericardial effusion developed in 327 patients; these 327 form our study group and represent 1.5% of all patients undergoing cardiac surgery within the 13-year time frame. This incidence was 1.5% in 1993 through 1999 and 1.8% in 2000 through 2005 ($p = 0.26$). The remaining 19,632 patients presumably did not have effusion (control 1); a subset of 10,158 patients without effusion had postoperative echocardiographic examination (control 2).

The mean (SD) age of patients in the study group was 62 (14) years (range, 18 to 92 years), and most were men (224, 69%). Characteristics of the study group and the 2 control groups are presented in Table 2. Patients with pericardial effusion were slightly younger and more overweight (mean body mass index, 29.2 kg/m^2) than those in the control groups, often had valvular heart disease (84%), and generally exhibited normal preoperative left ventricular ejection fraction (mean, 0.55). Of importance, the majority of patients without pericardial effusion and echocardiographic examination after surgery (control 2) had valvular operations (90%).

Manifestations of Pericardial Effusion

Pericardial effusion was commonly visualized by echocardiography at the end of the first postoperative week (mean [SD], 9 [6] days; median, 7 days). In 140 patients (43%) pericardial effusion was initially small, which progressed further to moderate or large in 81 patients (25%). Moderate or large pericardial effusion was usually seen approximately 2 weeks after the operation (13 [6] days).

For 280 patients (86%) with pericardial effusions, some, mostly nonspecific, symptoms were reported in the medical records; 47 patients (14%) had no reported problems.

Table 3. Symptoms and Signs of Pericardial Effusion (n = 327)

Symptom or Sign	Number of Patients (%)
Symptoms	
Dyspnea	116 (35)
Malaise/fatigue	71 (22)
Chest pain/discomfort	43 (13)
Edema	32 (10)
Presyncope/syncope	26 (8)
Nausea/vomiting	21 (6)
Abdominal pain	6 (2)
Signs	
SBP <90 mm Hg	91 (28)
HR >100 bpm	87 (27)
Oliguria	38 (12)
Fever (>38°C)	35 (11)
Increased JVP	40 (12)
Decreased cardiac output	21 (6)
Paradoxical pulse	22 (7)
Cardiac arrest	14 (4)

bpm = beats per minute; HR = heart rate; JVP = jugular venous pressure; SBP = systolic blood pressure.

Dyspnea and malaise occurred most frequently (Table 3). Clinical features of tamponade including hypotension, tachycardia, and decreased cardiac output were documented in 138 study patients (42%), 94 of whom also had Doppler or 2D echocardiographic evidence of tamponade. Pericardial effusions were associated with pleural effusions in 179 cases (55%); 104 bilateral, 65 left-sided, and 10 right-sided pleural effusions were seen on chest radiography. Also, 215 study patients (66%) had an enlarged cardiac silhouette.

Transthoracic echocardiography was the main tool for diagnosis of pericardial effusion (221 patients). In 67 patients significant pericardial effusion or tamponade was diagnosed clinically and confirmed by echocardiography. Pericardial effusion was revealed by computed tomography in 24 patients and was suspected on the basis of chest radiography and further confirmed by echocardiography in 11 patients. In 4 patients, diagnosis of effusion was made at the time of emergent exploratory sternotomy for cardiac tamponade.

The mean (SD) size of the echolucent space in the pericardium of study patients was 30 (12) mm (range, 7 to 70 mm). In 135 patients (41%) pericardial effusion was described as moderate-large or large (≥ 25 mm). Pericardial effusion was commonly circumferential (n = 117, 36%). Evidence of clot or hematoma in the pericardial space was seen in 34 patients (10%). For 198 patients (61%), Doppler or 2D echocardiography showed evidence of tamponade; 94 patients, as mentioned above, had clinical symptoms of severe hemodynamic compromise.

Eighty-six patients (26%) had small or small-moderate pericardial effusions that were considered clinically insignificant at the time of hospital dismissal. Subse-

quently, effusion progressed in 45 of these patients (52%), prompting readmission to the hospital for drainage (mean [SD] postoperative day, 16 [7]). Of interest, 66 of these 86 patients (77%) had valve surgery.

Management of Pericardial Effusion

The management strategy for pericardial effusion depended on the patient's clinical condition and the echocardiographic findings. Anticoagulation with warfarin or heparin was usually discontinued or the dose was decreased. If the international normalized ratio was high (>4.5), vitamin K and fresh frozen plasma were administered and drainage of the effusion was postponed for 1 or 2 days, if the patient remained stable hemodynamically, until the international normalized ratio decreased. Surgical or catheter drainage was used for all patients who had clinical evidence of tamponade. In patients without prominent symptoms or clinical findings, the decision to use drainage depended on Doppler-2D echocardiographic features of tamponade physiology and the amount and accessibility of the fluid. If an effusion was hard to reach by percutaneous drainage (eg, posterior location) and the clinical condition was stable, the patient was observed without intervention. In emergent situations caused by tamponade (cardiac arrest, ventricular fibrillation, or tachycardia with hemodynamic collapse), emergent resternotomy was performed.

In 169 patients (52%), pericardial effusions were evacuated by echocardiographically guided pericardiocentesis. Surgical drainage was performed in 75 patients (23%), and effusion was managed conservatively in 67 patients (20%). In 13 cases (4%), recurrent effusion required a staged approach (initial pericardiocentesis, followed by surgical drainage). Pericardial effusion resolved after left thoracocentesis for pleural effusion in 3 patients (1%).

Pericardiocentesis was preferred to surgical drainage if pericardial fluid was considered easy to reach and tap by catheter under echocardiographic guidance. Pericardiocentesis was performed more often after the seventh postoperative day (n = 144, 79% of 182 procedures). In most patients, extended pericardial catheter drainage was used (every 4 to 6 hours), which was usually removed 2 to 3 days after initial pericardiocentesis. Pericardiocentesis was effective in 97% of performed procedures; repeated drainage insertion was required in 4 cases (2%). One complication occurred after pericardiocentesis (suspicion of heart chamber perforation) with consequent surgical reexploration.

Surgical drainage of pericardial effusion was usually performed earlier after the initial operation, within the first 7 days after surgery in 50 of 88 procedures (57%); in most instances echocardiographic evidence of pericardial clot or hematoma or suspicion of ongoing bleeding was present. Of note, the source of bleeding was found in 12 patients (14%). Resternotomy for surgical drainage was performed in 83 cases (emergent resternotomy in the intensive care unit in 8 cases); a subxiphoid approach was chosen in 5 cases.

The mean amount of fluid evacuated by pericardiocentesis was 567 (356) mL and by surgical drainage was 648 (361) mL. The fluid samples were sent for microbiological analysis, which showed positive growth in 9 patients (3%).

Nonoperative treatment of pericardial effusions in 30 patients consisted of administration of diuretics, nonsteroidal antiinflammatory drugs, rarely corticosteroids (n = 8, 2%), or close observation only (n = 37) if the hemodynamic significance of the pericardial effusion was doubtful or the risks of performing pericardiocentesis were high because of the location of the effusion. For 24 patients treated conservatively, echocardiographic follow-up data beyond 1 week after diagnosis of pericardial effusion was available. Approximately 5 weeks (mean, 37 [29] days; median, 26 days) after initial diagnosis, the effusion completely resolved in 16 patients, decreased in 3 patients, and remained unchanged in 5 patients. No association was found between size of the effusion and outcome of nonoperative treatment; some small effusions persisted for more than 1 month, and some moderate effusions resolved in 2 weeks.

Table 4. Independent Risk Factors for Postoperative Pericardial Effusion; All Patients

Variable	Odds Ratio[a]	95% CI	*p* Value
BSA (increase by 1 unit)	3.1	2.0–4.8	<0.001
Pulmonary thromboembolism	6.7	2.8–16.0	<0.001
Renal failure	1.6	1.1–2.4	0.02
Immunosuppression	2.0	1.4–2.9	<0.001
CAD	0.7	0.6–1.0	0.02
Type of operation[b]			
Heart transplant	9.5	5.2–17.7	<0.001
Aortic aneurysm	4.3	2.1–8.9	<0.001
Valve	3.7	2.6–5.2	<0.001
VAD	2.7	1.1–6.5	0.03
Surgery status, urgent/emergent[c]	1.6	1.2–2.1	0.001
CPB time (increase by 10 min)	1.1	1.0–1.1	<0.001

[a] For 327 patients with pericardial effusion versus 19,632 patients without effusion; area under the curve, 0.744; Hosmer-Lemeshow test $p = 0.56$. [b] Odds ratios calculated using coronary artery bypass grafting as the reference. [c] Odds ratio calculated using elective surgery as the reference.

BSA = body surface area; CAD = coronary artery disease; CI = confidence interval; CPB = cardiopulmonary bypass; VAD = ventricular assist device implantation.

Risk Factors for Pericardial Effusion

Patient characteristics considered possible risk factors for pericardial effusion (Table 1) were compared between the 327 patients with effusion and 19,632 patients without effusion (control 1). On univariate analysis, 26 of 41 variables included in the analysis appeared to be significantly different between the 2 groups. The multivariate logistic regression model showed increased body surface area, immunosuppression, pulmonary thromboembolism, renal failure, urgency of operation, type of operation (heart transplant, valve, aortic aneurysm surgery, ventricular assist device insertion), and prolonged cardiopulmonary bypass to be independent risk factors for development of pericardial effusion (Table 4).

Table 5. Independent Risk Factors for Postoperative Pericardial Effusion; Study Patients and Control 2 Group

Variable	Odds Ratio[a]	95% CI	*p* Value
BSA (increase by 1 unit)	3.1	1.9–4.8	<0.001
Hypertension	1.4	1.1–1.8	0.004
Immunosuppression	2.1	1.5–3.1	<0.001
Pulmonary thromboembolism	4.3	1.8–10.4	0.001
Previous operations with CPB	0.5	0.3–0.7	<0.001
Type of operation[b]			
Valve	0.8	0.6–1.0	0.07
Aortic aneurysm	2.9	1.4–5.9	0.004
Heart transplant	2.8	1.6–4.9	0.001
Other[c]	0.5	0.3–0.9	0.01
CPB time (increase by 10 min)	1.1	1.0–1.1	<0.001

[a] For 327 patients with pericardial effusion vs 10,158 patients without effusion and postoperative echocardiographic examination; area under the curve, 0.690; Hosmer-Lemeshow test $p = 0.32$. [b] Odds ratios calculated using coronary artery bypass grafting as the reference. [c] Myectomy, maze procedure, tumor excision, postinfarction ventricular septal defect repair.

BSA = body surface area; CI = confidence interval; CPB = cardiopulmonary bypass.

The second logistic regression model included the data of our study cohort (n = 327) and 10,158 patients who had no effusion and underwent postoperative echocardiographic evaluation (control 2). On univariate analysis, 30 of 41 variables included were significantly different between the 2 groups. In the multivariate model of this second analysis, predisposing risk factors for effusion were similar to those from the first analysis (Table 5). An additional finding was that patients having previous cardiac operations had a lower risk of effusion. Valve surgery was not a significant risk factor in this second analysis.

Comment

The reported incidence of postoperative pericardial effusions ranges between 1% and 77%, depending on study definitions and designs [3–8]. The higher incidences are reported from smaller prospective studies with frequent echocardiographic examinations; most of these effusions were clinically insignificant [5–7]. In retrospective studies of clinically important pericardial effusions the reported incidence is 1% to 2% [3, 4, 8]. In the current study, the incidence of observed pericardial effusion was relatively low (1.5%), probably because it was defined as fluid in the pericardial space that required intervention, lengthened in-hospital stay, or was large enough to prompt consideration of drainage. Therefore, cases of effusion that did not meet the above criteria or were not detected (due to asymptomatic course and absence of routine echocardiographic follow-up after certain types of operations, such as coronary artery bypass grafting) were not included in the study. Despite the introduction of new protocols for anticoagulation and new antiplatelet drugs during the study period, we found no significant change in the incidence of postoperative effusions from the beginning to the end of the study period.

Symptoms of pericardial effusion are nonspecific, and few patients have the classic presentation of tamponade; indeed, many patients with pericardial effusion report minimal problems [1, 4]. In the present study, 42% of patients manifested hemodynamic compromise with hypotension, low cardiac output, and (or) oliguria. Other signs of tamponade were reported infrequently, but echocardiographic evidence of tamponade was found in 61%. Therefore, the threshold for performing an echocardiographic study after cardiac surgery if atypical symptoms are present should be reasonably low.

A group of patients in our study cohort were dismissed from the hospital after valve surgery with small clinically insignificant pericardial effusions; 52% of them were readmitted because of tamponade approximately 2 weeks after surgery. It is important to closely monitor this category of patients after dismissal and educate the patients and relatives to be alert to possible nonspecific symptoms.

Large pericardial effusions require drainage in most cases, and the choice between open drainage and pericardiocentesis is based on location and character of the effusion, as well as physician preference and experience. At Mayo Clinic, echocardiography-guided pericardiocentesis has been performed for drainage of postoperative pericardial effusions since the 1980s [9]. In the current study this technique was used for 56% of patients who had initial drainage of effusion, and was almost always successful. Effectiveness of initial pericardiocentesis was enhanced by leaving a pigtail catheter in the pericardial space for subsequent aspirations. The safety of echocardiography-guided pericardiocentesis has been documented by others [3, 5, 9, 10].

Few studies have analyzed the risk factors for development of postoperative pericardial effusions. Most of the previous investigations focused on effusions after heart transplant [7, 11–14]. Multivariate analysis in the study of Al-Dadah and colleagues [11] failed to identify risk factors for pericardial effusions in patients undergoing heart transplant other than prolonged donor ischemic time. Vandenberg and colleagues [12] reported that a combination of 3 risk factors (preoperative diagnosis of dilated cardiomyopathy, cyclosporine therapy, and acute rejection) yielded an 86% probability of pericardial effusion development in their group of heart transplant patients. Hauptman and colleagues [14] reported that a positive mismatch in weight between recipient and donor was a risk factor for pericardial effusion. The absence of prior cardiac operations was found to be "the most powerful predictor of effusion formation" [14].

In our study, heart transplant was an independent risk factor for pericardial effusions. This association likely results from a combination of factors including the potential space created when an enlarged, diseased heart is replaced with a normal organ and effects of immunosuppression, which may inhibit obliteration of serosal surfaces. This hypothesis is consistent with another major

finding, namely that preoperative administration of immunosuppressive medications, including corticosteroids and cytostatics, increased the risk of pericardial effusion. Most patients who received immunosuppressive medications preoperatively had comorbid conditions (eg, rheumatoid arthritis, systemic lupus erythematosus, or tumors), and these patients might be predisposed to coagulopathy and impaired immune response postoperatively.

Our study also showed a lower probability of postoperative pericardial effusions in patients who underwent prior operations with cardiopulmonary bypass. There are several possible explanations for lower observed incidence of effusion in patients having reoperation. In some circumstances, such as reoperation for aortic valve replacement, the surgeon may not completely free the heart from pericardial adhesions, and the resulting pericardial space is smaller than in patients having primary operations. Also, development of late effusions may be decreased because the serosal surface is not intact.

Pericardial effusions were frequently accompanied by pleural effusions; this also has been reported by Ikäheimo and colleagues [4], who suggested that patients with persistent pericardial effusions after cardiac operations were more likely to have persistent pleural effusions. Bilateral pleural effusions, unlike left-sided effusions, were more likely to be related to heart failure or an inflammatory reaction to operative trauma (topical cardiac cooling or pleurotomy) than to postoperative bleeding.

The finding that valve surgery was not a significant risk factor in the analysis of the study group versus the control 2 group may be explained by the fact that the control 2 group had more patients undergoing valve surgery (79%), whereas in the control 1 group, valve surgery was performed in 47% of cases. Our study cohort also had a high percentage of patients with valve surgery (68%), so the importance of this risk factor could not be adequately estimated in the second model. However, based on the results of the first multivariate analysis, we believe that valve surgery is a significant factor predisposing patients to pericardial effusion.

Observed incidence of postoperative effusions was lowest among patients who had coronary artery bypass grafting, possibly because the left pleural space was opened for harvest of the internal mammary artery. Of note, because not all patients undergoing coronary artery bypass grafting had postoperative echocardiography, the lower incidence of effusions in this group can be attributed to lower probability of the effusion being revealed.

Aortic aneurysm surgery was an independent risk factor for postoperative pericardial effusions, according to our analysis. Other reports have described the same finding; aortic root surgery is associated with increased risk of pericardial effusions [15, 16]. One possible explanation for this phenomenon is a greater degree of early postoperative bleeding leading to more retained mediastinal clot, which may in turn lead to greater local inflammation. Furthermore, as this clot lyses it may be expected to promote osmotic accumulation of fluid in the perigraft space. Although data on chest tube drainage were not available for all patients, it is likely that patients who bleed excessively after operation (including those undergoing reoperation) are more likely to have retained thrombus. A second potential mechanism is late serous "weeping" through the graft material itself. Finally, aortic procedures often require significant mediastinal dissection, which may disrupt lymphatics.

Postoperative pericardial effusions were more likely to develop in patients with renal failure and prolonged bypass time. This supports the association between pericardial effusion and fluid retention in serous cavities as well as the possible association with systemic inflammatory response.

On the basis of clinical experience one would expect an association between anticoagulation therapy and pericardial effusions. However, in most publications the association of anticoagulant use with increased risk of pericardial effusion is based only on the documented administration of anticoagulants in a large proportion of patients with effusions, and few studies report anticoagulation status of control patients without effusions [5, 8, 17]. In an echocardiographic study, Malouf and colleagues [6] compared the incidence of pericardial effusions in postoperative patients who did and did not receive systemic anticoagulation. In that report, the incidence of small and moderate pericardial effusions was similar between the 2 groups, but the incidence of large effusions was significantly higher in patients who received anticoagulation [6]. In our study, history of pulmonary thromboembolism and valve surgery were independent predictors of pericardial effusions, which were directly associated with the disturbances in coagulation. For a better understanding of the nature of pericardial effusions in these subgroups, more focused analysis of coagulation profiles and administered anticoagulants is required.

Study Limitations

Pericardial effusion was defined as fluid in the pericardial space that manifested with symptoms, required specific treatment, was large enough to prompt consideration of drainage, or required lengthened in-hospital stay for observation. Patients who did not meet the above criteria, required reexploration for bleeding or tamponade within the first 3 days after surgery, or did not provide research authorization were excluded. This is one potential limitation of the study. Patients younger than 18 years were not included in this study, although other publications have demonstrated the incidence of pericardial effusion after surgery to be high in young patients with congenital pathology [7]. Because postoperative echocardiographic evaluation was not performed in all control patients without effusion in this study, the conclusion that these patients did not have effusion is somewhat presumptive. To decrease this bias we identified the subgroup of control 2 patients who had postoperative echocardiography; however, this patient subset was not homogeneous because patients were not selected randomly, so this group contained more patients with valve surgery. Nevertheless, the results of our first and second risk factor

analyses were very similar. In addition, we evaluated the reliability of the multivariate models; thus, we are confident that the study results are accurate.

Conclusion

Several groups of patients are at increased risk for postoperative effusions. Patient care protocols, especially those concerning echocardiographic surveillance, should take these findings into consideration. Patients dismissed from the hospital with small clinically insignificant pericardial effusions, especially after valve surgery, should be educated as to the risk of progression of effusions (52% in this study) and the attendant nonspecific symptoms such as fatigue and dyspnea.

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